



DEPARTMENT OF THE AIR FORCE
59TH MEDICAL WING (AETC)
JOINT BASE SAN ANTONIO - LACKLAND TEXAS

26 JAN 2017

MEMORANDUM FOR SGOZ

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Linda Steel-Goodwin

LINDA STEEL-GOODWIN, Col, USAF, BSC
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A Case of Chagas Cardiomyopathy Following Infection in South Central Texas

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DISCLAIMER

The views expressed are those of the authors and do not reflect the official views or policy of the Department of Defense and its Components. This case report was reviewed by the 59th Medical Wing Institutional Review Board (FWH20170019N) and determined to be non-research.

ABSTRACT

Nearly 8 million people globally are infected with *Trypanosoma cruzi*, the causative parasitic agent of Chagas disease. The vast majority of incident infections originate in pockets of Latin America endemic to the parasite and its vector, the triatomine insect. Since 1955, when the first locally-acquired case was reported, there have been fewer than 30 autochthonous cases documented in the United States. We describe the case of an 18 year-old U.S. Air Force trainee, a native Texan with no travel history beyond the continental United States, who screened positive for *T. cruzi* infection on blood donation and was subsequently found to have chronic Chagasic cardiomyopathy. This is the first documented case of Chagas disease in a U.S. military trainee and one of the first known autochthonous cases of Chagasic cardiomyopathy in a Texas resident. Diagnostic, therapeutic, and military implications are discussed.

BACKGROUND

Human Chagas disease is caused by the protozoan parasite *Trypanosoma cruzi*, acquired primarily through contact with infected excreta of triatomine insects (known colloquially as “kissing bugs”). Although vector-borne transmission predominates, humans can also become infected congenitally, orally through contaminated food or beverages, or hematogenously through blood transfusion or organ transplantation.¹ During the first 4-8 weeks of infection, considered the acute phase, symptoms are usually mild, nonspecific, or unappreciable; potentially fatal myocarditis or meningoencephalitis occur rarely. Approximately 70-80% of infected persons enter a chronic indeterminate phase, characterized by lifelong infection without symptoms, electrocardiographic changes, and radiographic evidence of disease. The remaining 20-30% develop clinical disease with cardiac and/or digestive manifestations, often presenting years or decades after infection.²

With nearly 8 million people infected worldwide, Chagas is classified by the World Health Organization as one of the most neglected tropical diseases.¹ There are an estimated 240 thousand prevalent cases in the United States among immigrants from endemic areas of Latin America.³ Fewer than 30 locally-acquired infections have been reported in the United States since 1955,⁴ when a resident of Corpus Christi, Texas, became the first documented autochthonous case in the country.⁵ In 2013, the Texas Department of State Health Services added Chagas disease to the state's Notifiable Conditions list, which requires the reporting of confirmed and suspected human cases to local or regional health departments. Twelve autochthonous human infections were confirmed in the first two years of mandatory reporting,⁶ at least one of which was associated with left ventricular dysfunction.⁷

CASE REPORT

In October 2016, an 18 year-old U.S. Air Force trainee screened positive for *T. cruzi* infection when he donated blood at Joint Base San Antonio (JBSA), Texas. Blood from all first-time donors at the JBSA-Lackland donation center is screened for *T. cruzi* with an enzyme-linked immunosorbent assay from Ortho-Clinical Diagnostics. Per standard protocol, he was referred to the installation's trainee health clinic, where he was found to have normal vital signs and an unremarkable physical exam. He reported being in excellent health and had not experienced any recent chest pain, shortness of breath, dizziness, or gastrointestinal symptoms.

A chemiluminescent immunoassay and enzyme strip assay (Abbott Laboratories) were ordered and found to be positive for anti-*T. cruzi* IgG antibodies. A whole blood sample was sent to the Reference Diagnostic Laboratory at the Centers for Disease Control and Prevention for further testing. An enzyme immunoassay was reactive and TESA immunoblot was positive, confirming the diagnosis. The patient was notified of these results and referred to the Department of Infectious Disease at San Antonio Military Medical Center (SAMMC) for further evaluation and treatment.

After notifying the patient of his laboratory results, Infectious Disease conducted a 12-lead electrocardiogram (ECG), which demonstrated normal sinus rhythm, first-degree atrioventricular block, and left anterior hemi-block with right bundle branch block. This prompted referral to the Division of

Cardiology at SAMMC. Cardiovascular physical exam was benign with normal heart sounds, normal jugular venous pressure, normal apical impulse, and no evidence of congestive heart failure. A battery of noninvasive tests was performed in order to assess for common cardiac manifestations of Chagas disease—including, but not limited to, left ventricular dilatation and dysfunction, wall motion abnormalities, aneurysms, diastolic dysfunction, pathologic bradyarrhythmias and tachyarrhythmias, and ischemic heart disease.⁸

The majority of tests were within normal limits. Chest x-ray showed no evidence of cardiomegaly. Holter monitoring was negative for any pathologic dysrhythmias. Transthoracic echocardiogram demonstrated normal diastolic, valvular, and global systolic function. Exercise testing with Bruce protocol established no exercise-induced arrhythmias, ischemic electrical changes, or anginal symptoms. Cardiopulmonary exercise testing found an appropriate VO₂ max, early anaerobic threshold, and normal VE/VCO₂ slope, consistent with a subclinical reduction in exercise capacity with preserved ventilatory efficiency. Cardiac magnetic resonance imaging confirmed the diagnosis of early heart disease demonstrating left ventricular cavity dilation with preserved global systolic function (ejection fraction of 76%); the imaging was otherwise normal with no wall motion abnormalities, late gadolinium enhancement, abnormal T1 relaxation, or myocardial edema on T2 weighted images.

Given his exposure history, serologic findings, abnormal ECG, and left ventricular cavity dilation, the patient was determined to have chronic Chagasic cardiomyopathy. Per the Brazilian Consensus Classification and American College of Cardiology/American Heart Association classification schemes, he was classified as Stage B1 and Stage B, respectively,^{9,10} and at low risk for cardiac death according to two validated risk calculators.^{10,11} Since cardiomyopathy is a disqualifiable condition for accession into the U.S. military,¹² the patient was processed for medical discharge from training. Infectious Disease advised the patient to complete a 60-day regimen of oral benznidazole,¹³ but he declined. He was strongly encouraged to seek follow-up in the civilian health care sector and to notify household contacts that they should be screened for Chagas disease.¹⁴

Public health personnel interviewed the patient to facilitate case reporting to the Texas Department of State Health Services. The patient was raised on a ranch in south-central Texas and had never traveled outside the continental United States. He camped occasionally near his home but never hunted or skinned animals. When shown a display case with triatomine insects of various species and at different stages of development, the patient immediately recognized them, saying they “were all over the place,” including within the residence. He did not recall ever receiving a bite. A number of reservoir animals were also present at the residence, to include raccoons, armadillos, cats, and dogs. The patient was not aware of any relatives having Chagas disease, although he was adopted at a young age and had no knowledge of his biological mother. He had never received a blood transfusion. A week before his blood donation he had spent five days and four nights on the JBSA Medina Training Annex for a field training exercise, during which he slept in a permethrin-treated bed net and reported no known insect bites.

COMMENT

Although neither congenital acquisition nor vector-borne acquisition during military training can be definitively ruled out, this patient was likely infected with *T. cruzi* while growing up on a ranch in south-central Texas. Ecologic modeling has predicted that this region of the United States is at increased risk for autochthonous Chagas disease.¹⁵ Situated at the interface of tropical and temperate biomes, south-central Texas has a number of environmental and cultural factors that may facilitate human exposure to *T. cruzi*: a diverse array of wildlife reservoirs and indigenous triatomine species; the popularity of high-risk outdoor activities, especially hunting and camping; and scattered colonias (impoverished, primarily Hispanic communities). As compared to modern urban and suburban houses, poorly constructed ranches, cabins, and colonias are more susceptible to colonization by triatomine insects and wildlife reservoirs, thus increasing the likelihood of human exposure to infected vectors.¹⁶

The southern United States is inhabited by 11 recognized species of triatomine insects, most of which are competent *T. cruzi* vectors and likely to be involved in enzootic transmission cycles among indigenous wildlife reservoirs.¹⁷ All species exist as nest parasites that opportunistically feed on a variety of vertebrate hosts, including humans. The capacity of a given species to transmit *T. cruzi* to humans is

largely dependent upon their distribution in the environment, capacity for dispersal, propensity to invade human dwellings, and feeding-to-defecation interval.¹⁷⁻¹⁹

In south-central Texas, *Triatoma gerstaeckeri* insects have been found to readily enter human dwellings and feed upon humans and domestic animals.^{20,21} This medium to large triatomine species inhabits much of the Edwards Plateau and South Texas Brush Country between the 96th and 103rd parallels, the southeastern corner of New Mexico, and northeastern Mexico.²² The *T. cruzi* infection rate of this species may exceed 60% in south-central Texas,^{20,21} and adult insects often have detectable human blood in their midgut.²³

Although the case patient was likely infected prior to arrival at JBSA, this report highlights the risk for autochthonous Chagas disease in the southern United States and underscores the importance of preventing Chagas and other arthropod-borne diseases while training in endemic areas. Engineering controls should focus on reducing vegetation around military field sites—to the maximum extent possible without disrupting the training mission—in order to decrease vector habitats. Administrative controls emphasizing site cleanliness should help minimize the population of woodrats, an important reservoir animal.²⁴ Finally, the four components of optimal personal protection should be meticulously employed: a properly-worn field uniform (i.e., sleeves rolled down, wrist openings secured, undershirt tucked into the pants, and pant legs tucked into the boots); permethrin treatment of the uniform blouse and pants; the application of either DEET-based (20%-40% concentration) or picaridin-based (20% concentration) insect repellent to exposed skin; and sleeping in a permethrin-treated bed net.²⁵ Finally, diligent public health surveillance and health care provider education for Chagas disease is warranted.

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